

Manual vibration increases expiratory flow rate via increased intrapleural pressure in healthy adults: an experimental study

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Question: What is the relationship between vibration of the chest wall and the resulting chest wall force, chest wall circumference, intrapleural pressure, and expiratory flow rate? Is the change in intrapleural pressure during vibration the sum of the intrapleural pressure due to recoil of the lung, chest wall compression, and chest wall oscillation? **Design:** Randomised, within-subject, experimental study. **Participants:** Seven experienced cardiopulmonary physiotherapists and three healthy adults. **Intervention:** Vibration (compression + oscillation), compression alone, and oscillation alone were applied manually to the chest walls of healthy participants during passive exertion and compared with passive expiration alone. **Outcome measures:** Chest wall force, chest wall circumference, intrapleural pressure, and expiratory flow rate. **Results:** During vibration, coherence was high ($r^2 > 0.97$) between external chest wall force, chest wall circumference, intrapleural pressure, and expiratory flow. The mean change in intrapleural pressure during vibration was 9.55 cmH₂O (SD 1.66), during chest compression alone was 8.06 cmH₂O (SD 1.65), during oscillation alone was 7.93 cmH₂O (SD 1.57), and during passive expiration alone was 6.82 cmH₂O (SD 1.51). During vibration, compression contributed 13% of the change in intrapleural pressure, oscillation contributed 12%, and lung recoil contributed the remaining 75%. **Conclusions:** During vibration the chest behaves as a highly linear system. Changes in intrapleural pressure occurring during vibration appear to be the sum of changes in pressure due to lung recoil and the compressive and oscillatory components of the technique, which suggests that all three components are required to optimise expiratory flow. [McCarren B, Alison JA, Herbert RD (2006) Manual vibration increases expiratory flow rate via increased intrapleural pressure in healthy adults: an experimental study. *Australian Journal of Physiotherapy* 52: 267–271]

Key words: Vibration, Maximal Expiratory Flow Rate, Chest Wall Oscillation

Introduction

Vibration is a therapy used widely to assist with the removal of secretions from the lungs of people with airways disease (McCarren et al 2003). Manual vibration is a combination of compression and oscillation of the chest wall during expiration (McCarren et al 2006).

It has been proposed that the forces applied to the chest wall during vibration are transmitted to the lungs and airways where they increase intrapleural pressure which in turn increases expiratory flow rate thereby loosening secretions mechanically. Vibration moves secretions towards the oropharynx (Kigin 1981, Opie and Spalding 1958) and increases expiratory flow rate in healthy participants (McCarren et al 2006). Animal and *in vitro* studies have shown that mechanical vibration increases expiratory flow rate (King et al 1983, King et al 1984), alters mucus rheology (King et al 1983, Tomkiewics et al 1994), and increases mucus transport (Gross et al 1985, King et al 1983). However the dynamics of therapeutic vibration have not been quantified in humans. Of particular interest is whether, given the impedance of the respiratory system (Peslin et al 1986), the high frequency oscillation component of vibration influences intrapleural pressure and expiratory flow rate. The primary aim of the current study was, therefore, to quantify the relationship between the forces applied to the chest wall, chest wall circumference, intrapleural pressure, and expiratory flow rate during vibration.

We have recently shown that the expiratory flow rate produced by vibration is approximately equal to the sum of the flow rates due to recoil of the lung and due to the compression and oscillation components of vibration (McCarren et al 2006). Therefore, the secondary aim of the current study was to determine if a similar relationship existed with intrapleural pressure. That is, we sought to determine if the intrapleural pressure experienced during vibration was the sum of the intrapleural pressure due to recoil of the lung, chest wall compression, and chest wall oscillation. This information will provide physiotherapists with an understanding of the factors that contribute to the effective application of vibration.

Method

Design The design was a randomised, within-subject, experimental study. Experienced cardiopulmonary physiotherapists manually applied vibration (compression + oscillation), compression alone, and oscillation alone to the chest walls of healthy participants. The resultant net chest wall forces, chest wall circumferences, intrapleural pressures, and expiratory flow rates were compared with those from passive expiration alone. The order of the interventions was randomised. The experimental protocol was approved by The University of Sydney Human Ethics Committee and informed consent was obtained from each participant.

Participants Three healthy adults (one male) with a mean

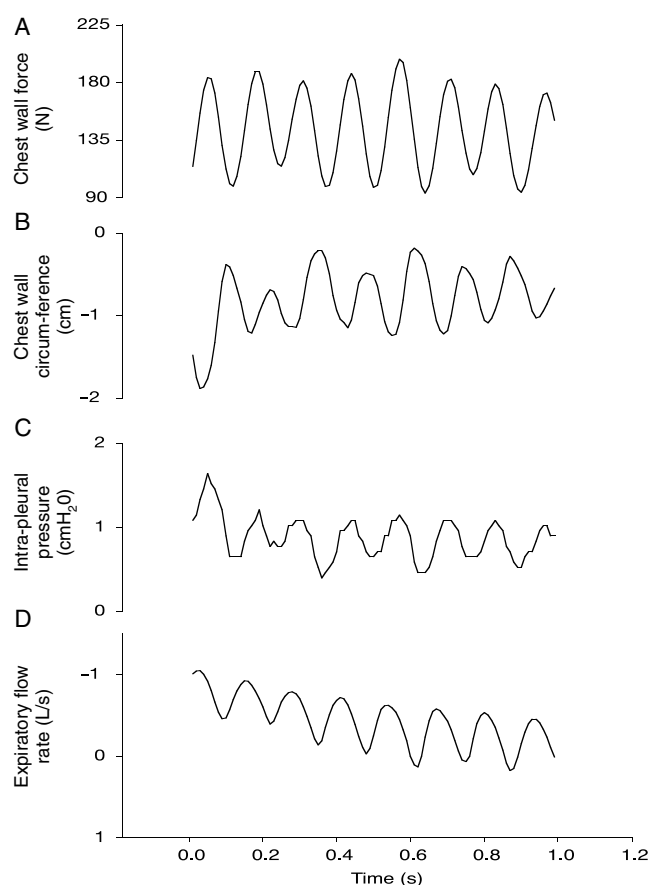


Figure 1. An application of vibration during the expiratory phase and the effect on (a) chest wall force, (b) chest wall circumference, (c) intrapleural pressure and (d) expiratory flow rate. Note that the axis of the expiratory flow rates is reversed to aid visualisation of the data, ie, more negative value = faster expiratory flow rates

age of 46.3 years (SD 4.9) and BMI of 22.6 kg/m² (SD 3.3) participated. They were non-smoking with normal respiratory function and no history of recent respiratory tract infections. The interventions were applied by seven physiotherapists, six females and one male with a mean of 13 years (SD 10.6) of clinical experience of which 11.5 years (SD 10.6) were of cardiopulmonary experience.

Intervention During four interventions, the healthy participants were requested to inspire to total lung capacity and expire passively. With the participants in left side-lying, the physiotherapists manually applied vibration (chest wall compression plus oscillation), chest wall compression alone, and chest wall oscillation alone. The physiotherapists were asked to apply chest wall compression alone and chest wall oscillation alone replicating the forces they would use during their application of vibration (McCarren et al 2006). These three interventions were compared to a control intervention of passive expiration from total lung capacity alone. Three trials of each intervention were performed.

Outcome measures Net chest wall force was measured with an instrumented bed (Chiradendjnant et al 2001). Seven strain-gauge load cells were mounted between the supporting surface and the bed frame, permitting three-dimensional characterisation of the external force applied to the chest wall.

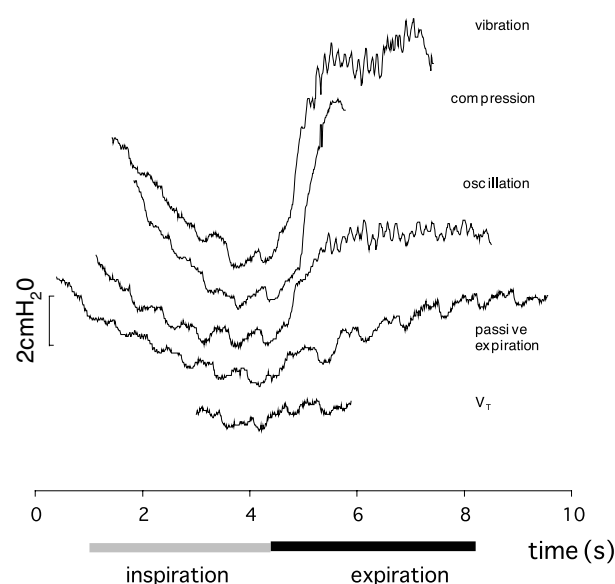


Figure 2. Intrapleural pressure during one respiratory cycle for each of the interventions in one participant. Downward deflections occur during the inspiratory phase and upward deflections during the expiratory phase. The five traces have been separated vertically for clarity. V_T = tidal breathing.

Chest wall circumference was measured with calibrated inductive plethysmography^(a). The root mean square error (accuracy) and frequency response of the respiratory inductive plethysmograph have been reported previously (McCarren et al 2006).

Intrapleural pressure was measured as oesophageal pressure, which reflects intrapleural pressure (Milic-Emili et al 1964), in order to examine if force applied to the chest wall was transmitted to the lungs. Intrapleural pressure was measured with a swallowed oesophageal balloon catheter^(b) (frequency response > 20 Hz, Hartford et al 1997) connected to a calibrated pressure transducer^(c) (frequency response 20 Hz). The balloon was 10 cm in length and was filled with 0.5 mL of air to minimise the effects of balloon volume on the oesophageal pressure (Milic-Emili et al 1964). The intrapleural pressure was checked to ensure that it was approximately -5 cmH₂O at the end of tidal expiration and slight adjustments were made to the position of the catheter to minimise artefacts due to cardiac oscillations.

Expiratory flow rate was measured with a heated pneumotachograph^(d) (frequency response >12 Hz). The pneumotachograph was calibrated before each experimental procedure using a 3 L syringe according to the recommended protocol (ATS/ERS Taskforce 2005). All data were sampled at 100 Hz.

Data analysis Net chest wall force, chest wall circumference, intrapleural pressure, and expiratory flow rate during expiration were determined. The change in intrapleural pressure was calculated as the intrapleural pressure at the end of inspiration minus the intrapleural pressure at the end of expiration. The peak intrapleural pressure was the maximal pressure achieved during expiration.

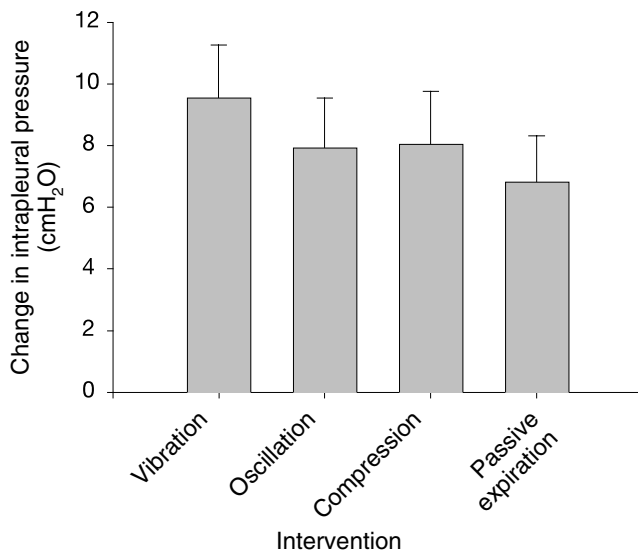


Figure 3. Mean (weighted average SD) change in intrapleural pressure (calculated as intrapleural pressure at end inspiration minus intrapleural pressure at end expiration) during the four interventions.

A cross-correlation analysis (Winter and Patla 1997) was carried out on the vibration data during the period when both compression and oscillation were being performed. We report the coherence (squared linear correlation), gain (amplification factor), phase (displacement), and frequency spectra (power) for frequencies of 0–15 Hz in increments of 0.5 Hz for the relationship between: chest wall force and chest wall circumference; chest wall circumference and intrapleural pressure; intrapleural pressure and expiratory flow rate; and chest wall force and expiratory flow rate. For all data, the mean and SD of the group was the weighted average of each experimental participant's means and SDs. The means were weighted by the number of physiotherapists (for the means) or the number of physiotherapists minus 1 (for the SDs) to account for the unequal number of physiotherapists who applied the interventions to each participant's chest. Coherence data were skewed, so the medians of these data are reported.

Results

Relationship between and chest wall force, circumference, intrapleural pressure and expiratory flow rate during vibration During vibration a strong linear relationship was apparent between chest wall force, chest wall circumference, intrapleural pressure, and expiratory flow rate. Figure 1 is a trial of one application of vibration. The group median coherence between chest wall force and chest wall circumference was 0.99, the group mean gain of the transfer function (the elastic stiffness of the chest wall) was 0.03 cm/N (SD 0.01), and the mean phase was 180 deg (SD 51). The median coherence between chest wall circumference and intrapleural pressure was 0.99, the mean gain was 0.92 cmH₂O/cm (SD 0.31), and the mean phase was 149 deg (SD 34). The median coherence between intrapleural pressure and expiratory flow rate was 0.97, the mean gain was 0.49 L/s/cmH₂O (SD 0.15), and the mean phase was 220 deg (SD 43). Finally, the median coherence between

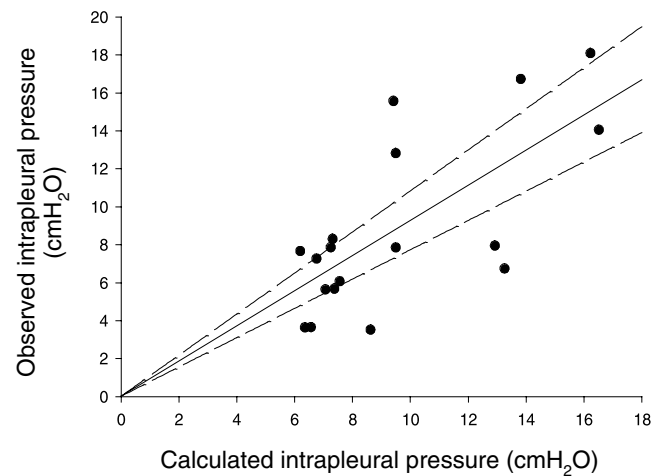


Figure 4. The relationship between observed and calculated mean change in intrapleural pressure (Δ IPP) during vibration ($n = 18$). The solid line is the regression line and the dashed lines are 95% CI. The regression line was forced through the origin. The relationship is described by the equation: observed Δ IPP = 0.93 (95% CI = 0.77 to 1.09) \times calculated Δ IPP ($r = 0.71$, $p < 0.001$).

chest wall force and expiratory flow rate was 1.00, the mean gain was 0.01 L/s/N (SD 0.01), and the mean phase was 237 deg (SD 39). See Tables 1 to 4 on the eAddenda for the complete dataset. In summary, there is a near-perfect linear relationship between force, change in chest wall circumference, intrapleural pressure, and expiratory flow during application of vibration.

Contribution of intrapleural pressure due to compression, oscillation and passive expiration to vibration A representative recording of the time course of the intrapleural pressure during each of the interventions is shown in Figure 2. The mean peak intrapleural pressure during vibration was 0.73 cmH₂O (SD 0.89), during compression alone was 0.26 cmH₂O (SD 1.01), during oscillation alone was -0.61 cmH₂O (SD 0.72), and during passive expiration alone was -1.37 cmH₂O (SD 0.48). The mean change in intrapleural pressure during vibration was 9.55 cmH₂O (SD 1.66), during compression alone was 8.06 cmH₂O (SD 1.65), during oscillation alone was 7.93 cmH₂O (SD 1.57) and during passive expiration alone was 6.82 cmH₂O (SD 1.51). The compression and oscillation components of vibration together increased intrapleural pressure, and therefore expiratory flow rate, by about one-third compared with those achieved during passive expiration alone. The change in intrapleural pressure during vibration was 16% greater than during compression alone, 18% greater than during oscillation alone, and 40% greater than during passive expiration alone (Figure 3).

The change in intrapleural pressure (Δ IPP) associated with manual vibration should be the sum of the changes in intrapleural pressure produced by the compression component and the oscillation component and lung recoil. That is:

$$\Delta \text{IPP}_{\text{vibration}} = (\Delta \text{IPP}_{\text{compression}} - \Delta \text{IPP}_{\text{passive}}) + (\Delta \text{IPP}_{\text{oscillation}} - \Delta \text{IPP}_{\text{passive}}) + (\Delta \text{IPP}_{\text{passive}}).$$

This model was supported by the observed data. Figure 4 shows the strong relationship between the calculated and observed data ($r = 0.71, p < 0.001$). During vibration in these healthy participants, lung recoil contributed approximately 75% of the change in intrapleural pressure, compression contributed 13%, and oscillation contributed the other 12%.

Discussion

This study shows that during vibration in healthy participants the net chest wall force, including the high frequency oscillation force, is transmitted to the lungs. The force applied to the chest wall, chest wall circumference, intrapleural pressure, and expiratory flow rate all changed in parallel with each other.

Relationship between and chest wall force, circumference, intrapleural pressure and expiratory flow rate during vibration This study is the first to examine the dynamics of manual vibration applied to humans. It shows there is a strong linear relationship between forces applied to the chest wall, displacement of the chest wall, intrapleural pressure, and expiratory flow rate during vibration. The phase indicated that during vibration the circumference of the chest wall decreased, intrapleural pressure increased, and this increased expiratory flow rate. The coherence indicated that 99% of the variance in flow was accounted for by the forces applied during vibration. This is consistent with the findings of studies that measure respiratory transfer impedance in response to mechanical vibration. During normal breathing (ie, passive expiration) expiratory flow rate is proportional to intra-alveolar pressure (recoil of the lung) and inversely proportional to airway resistance (Ward et al 1994). The high coherence between force applied to the chest wall and expiratory flow rate suggests that the additional forces applied to the chest wall during vibration by manual compression and oscillation add to the effects of lung recoil on expiratory flow rate. It also provides evidence for the proposal that this relationship increases mucus transport (King et al 1983, King et al 1990). The clinical implication of this finding is that both compression and oscillation of vibration are required to optimise the effects on expiratory flow and therefore secretion clearance.

During vibration, the gain of chest wall circumference (0.03 cm/N), intrapleural pressure (0.92 cmH₂O/cm), and expiratory flow rate (0.49 L/s/cmH₂O) are of participants with normal respiratory systems and not of patients with respiratory disorders. In patients with restrictive chest walls or those who are paralysed and anaesthetised we would predict that the elastic stiffness of the chest wall would be different. In addition, vibration is often used for patients who have excessive secretions (McCarren et al 2003), usually accompanied by airway inflammation and increased airway resistance (West 1987). The resultant expiratory flow would be predicted to be reduced in the presence of increased airway resistance (Rodarte and Shardonofsky 2000). Therefore, the relationship between the application of vibration and intrapleural pressure and expiratory flow rate may be reduced in patients with excessive secretions. The question remains as to whether vibration can still cause enough of an increase in expiratory flow rate to be effective in this case.

Contribution of intrapleural pressure due to compression, oscillation and passive expiration to vibration To our knowledge these are the first published data of intrapleural

pressure during manual vibration in humans. Other studies have reported the effects of mechanical vibration in humans (Fink and Mahlmeister 2002, Jones et al 1995) or in animals (Markhorst et al 2005), and manual vibration in animals (Wong et al 2003). In our healthy adults, forces applied to the chest wall during vibration, oscillation alone, and compression alone, were transmitted to the lungs. Vibration resulted in higher changes in intrapleural pressure than oscillation alone, compression alone, or passive expiration alone.

In conclusion, the chest behaves as a highly linear system during vibration; chest wall circumference, intrapleural pressure, and expiratory flow rate change in parallel with the net force applied to the chest wall. Changes in intrapleural pressure produced by vibration appear to be the sum of the effects of lung recoil and the compressive and oscillatory components of the technique.

eAddenda Tables 1–4 available at www.physiotherapy.asn.au/AJP

Footnotes ^(a)Respirace™, Ambulatory Monitoring Inc, Ardsly, NY, USA ^(b)Jaeger, Leibnizstra Hoechberg, Germany ^(c)Validyne DP 45, Northridge, CA, USA ^(d) Hans Rudolph Model 3813, Hans Rudolph Inc, Kansas City, Missouri

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